

Propranolol induced bradycardia in tetralogy of Fallot

DEBORAH J CLARK, K C CHAN, JOHN L GIBBS

From the Department of Paediatric Cardiology, Killingbeck Hospital, Leeds

SUMMARY When an 18 month old girl who had tetralogy of Fallot and episodes of severe cyanosis with loss of consciousness was treated with propranolol there was some improvement. But when the dose was increased she had further episodes of near syncope. Holter monitoring showed extreme intermittent bradycardia with pauses of up to 2.6 seconds. The episodes of near syncope and the bradycardia resolved after propranolol was stopped.

Apparent failure of propranolol treatment may on rare occasions be related to drug induced bradycardia rather than to continued episodes of severe cyanosis.

Propranolol is widely accepted as an effective palliation for tetralogy of Fallot complicated by episodes of severe cyanosis.¹ Side effects of propranolol, such as bradycardia, congestive heart failure, bronchospasm, and hypoglycaemia, are well recognised in adults but seem to be rare in infancy even at high doses.² We report the case of a child with tetralogy of Fallot who was treated with propranolol and in whom continued episodes of pallor and near syncope seemed to be caused by propranolol induced bradycardia.

Case report

An 18 month old Asian girl with tetralogy of Fallot had typical episodes of severe cyanosis during which she became distressed and then limp. Sometimes she lost consciousness for up to 10 minutes. She weighed 8 kg and she was treated with propranolol 5 mg three times a day. This seemed to abolish the attacks. During a subsequent outpatient visit, however, she had a prolonged episode of severe cyanosis and lost consciousness. She was admitted to hospital and the dose of propranolol was increased to 8 mg three times a day. After this she had several episodes each day when she became pale and suffered near syncope. Unlike her usual episodes these attacks were not associated with preceding cyanosis or distress. Because she had intermittent sinus bradycardia during a 12 lead electrocardiogram, 24 hour Holter monitoring was performed and β blockade was not

increased. She remained symptom free during the period of monitoring. Analysis of the recording showed sinus rhythm at a resting rate of 120 beats/minute with repeated daytime episodes of sinus bradycardia and sinus arrest resulting in extreme junctional bradycardia and pauses lasting up to 2.6 seconds (figure), followed by a slow junctional escape rhythm and recovery of normal sinus rhythm. The dose of propranolol was stopped and the child was sedated and nursed in a quiet room. After β blockade was stopped no further bradycardias occurred on continuous electrocardiographic monitoring and she remained free from episodes of severe cyanosis until she underwent corrective operation one week later. Her postoperative course was complicated by low cardiac output and she died three days after operation.

Discussion

Episodes of severe cyanosis may present with floppiness and pallor, transient vacant episodes, or convulsions.³ Although the exact mechanism of these episodes is uncertain, treatment with propranolol often abolishes them.^{4,5} Toxic effects are rare with the commonly used daily doses of between 2 and 5 mg/kg and doses of up to 14 mg/kg have proved both effective and free from side effects in children with supraventricular tachycardia.² Complications of treatment such as bradycardia, congestive heart failure, and somnolence have been reported in children, but these were rarely severe enough for treatment to be stopped.⁶ The paroxysmal sinus arrest

Requests for reprints to Dr John L Gibbs, Department of Paediatric Cardiology, Killingbeck Hospital, York Road, Leeds LS14 6UQ.

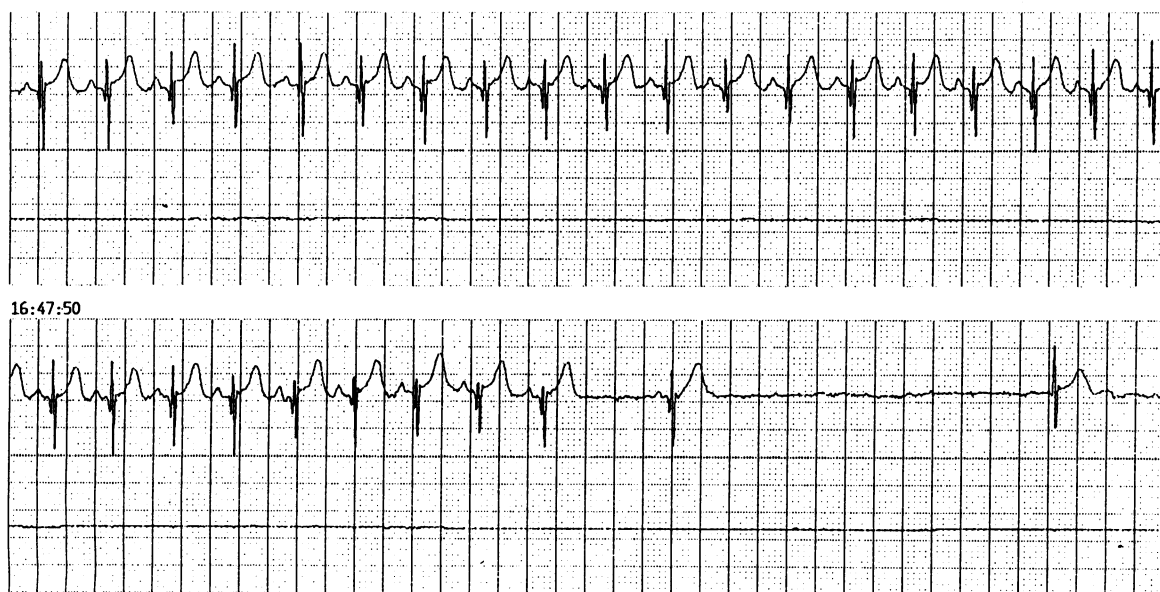


Figure Part of the Holter monitor recording taken during treatment with propranolol 3 mg/kg a day, showing abrupt sinus bradycardia followed by sinus arrest and a junctional escape beat after a pause of 2.6 seconds.

with episodes of extreme bradycardia that we saw in our patient has not been recognised before in a child treated with propranolol, irrespective of the dose given or the underlying cardiac abnormality.

Inadequate dosage may well be the most common cause of failure of episodes of severe cyanosis to respond to propranolol; an initial daily dose of 1 mg/kg has been suggested, gradually increased up to a maximum of 5 mg/kg and still further increased to 10 or 15 mg/kg if initial control of symptoms is lost.¹ While this protocol undoubtedly may prove successful in many children, our patient shows that potentially serious side effects may occur even with a standard dose and that continued symptoms in a child with tetralogy treated with propranolol may be related to iatrogenic bradycardia rather than to continued episodes of severe cyanosis.

References

- 1 Garson A, Gillette PC, McNamara DG. Propranolol: the preferred palliation for tetralogy of Fallot. *Am J Cardiol* 1981;47:1099-104.
- 2 Pickoff AS, Zies L, Ferrer PL, *et al.* High dose propranolol therapy in the management of supra-ventricular tachycardia. *Pediatrics* 1979;94:144-6.
- 3 Anderson RH, Macartney FJ, Shinebourne EA, Tynan M, eds. *Paediatric cardiology*. London: Churchill Livingstone, 1987:777.
- 4 Eriksson BO, Thorén C, Zetterqvist P. Long-term treatment with propranolol in selected cases of Fallot's tetralogy. *Br Heart J* 1969;31:37-44.
- 5 Ponce FE, Williams LC, Webb HM, Riopel DA, Hohn AR. Propranolol palliation of tetralogy of Fallot: experience with long term drug treatment in pediatric patients. *Pediatrics* 1973;52:100-8.
- 6 Gillette PC, Garson A, Eterovic E, Neches W, Mullins CE, McNamara DG. Oral propranolol treatment in infants and children. *J Pediatr* 1978;92:141-4.